

Guillain–Barré syndrome in *Plasmodium falciparum* malaria

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Summary: A patient with *Plasmodium falciparum* malaria developed peripheral neuropathy. Clinical, cerebro-spinal fluid examination and nerve conduction studies confirmed Guillain–Barré syndrome, not previously reported in *P. falciparum* malaria.

Introduction

Neurological complications such as cerebral malaria,¹ cranial nerve lesions, extrapyramidal tremor, transient paranoid psychosis and febrile convulsions in children are known to accompany *Plasmodium falciparum* malaria.² Cerebellar disturbances, frontal lobe syndrome and bulbar paralysis have also been reported,³ as have the periodic type of muscular paralysis⁴ and subarachnoid haemorrhage.⁵ I report here a case of Guillain–Barré syndrome following *Plasmodium falciparum* malaria in Sri Lanka.

Case report

A 45 year old farmer was admitted with a 3 day history of fever, chills and headache. On admission, his temperature was 39.8°C, he was conscious and rational, the liver and spleen were not palpable and the neck was supple. The central nervous system was normal on examination. There was no preceding sore throat, cold or cough. He did not drink alcohol. A clinical diagnosis of malaria was made. His haemoglobin was 12.2 g/dl, white cell count $8.8 \times 10^9/l$ with 80% neutrophils. A blood film showed asexual stage trophozoites of *Plasmodium falciparum*. Antimalarial therapy was commenced with choroquine and primaquine.

Two days after admission he complained of difficulty in walking together with numbness and weakness of his lower limbs. Examination revealed hypotonia of all four limbs; power was diminished

in the hips, knees and ankles to MRC grade 4.* The ankles were weaker than the hips. Power was also diminished in the shoulders, elbows and hands to MRC grade 4.* There was no fasciculation or wasting. Touch and pinprick sensation were impaired in the forearms and legs. Joint sense and vibration were absent in the toes. The ankle jerks were absent. All the other reflexes were decreased. Plantar response was flexor. Cranial nerves and funduscopy were normal. Further investigations showed fasting blood sugar 4.5 mmol/l, sodium 138 mmol/l, potassium 4.4 mmol/l and blood urea 5.2 mmol/l. Cerebrospinal fluid was clear in appearance, lymphocytes 2/mm³, proteins 120 mg/dl.

A diagnosis of acute polyneuritis was made and physiotherapy commenced. Over the next few days the weakness in the lower limbs progressed and power decreased to MRC grade 2.* His pulse and blood pressure remained stable. He did not develop any respiratory distress. His micturition was normal. At repeat lumbar puncture, the cerebrospinal fluid protein had risen to 600 mg/dl while the cell pattern remained unchanged.

Nerve conduction studies showed the following results. In the median nerve, latency was 8.5 m/s (range 3.2–4.2 m/s) while the conduction velocity was 22 m/s (range 50–67 m/s). In the ulnar nerve, latency was 6.1 m/s (normal 4.5 m/s), while velocity was 31 m/s (range 45–60 m/s).⁶ He showed signs of recovery after 3 weeks and gained normal power in his limbs after 6 weeks.

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*MRC grading: 4 = movement which is possible against gravity plus resistance but which is weaker than normal; 2 = movement which is possible with gravity eliminated.

Discussion

The neurological signs in this patient suggested lower motor neurone paralysis. The nerve conduction studies showed prolonged latency and slowing of conduction suggestive of demyelination. Elevated proteins and the absence of cells in the cerebrospinal fluid also suggest Guillain-Barré syndrome.

This clinical picture developed after an attack of *Plasmodium falciparum*. Therefore it is reasonable to assume a causal relationship although the aetiology of polyneuropathy in *P. falciparum* infections is not known.

The malaria parasite may damage peripheral nerves by vascular occlusion, thus causing anoxaemic stagnation in the vasa nervosum leading to temporary demyelination and recovery after disappearance of parasitaemia and establishment of normal blood flow in vasa nervosum.

Immune-mediated damage is believed to be the cause of Guillain-Barré syndrome. In malaria, asexual stage infection is accompanied by the release of cytokines and other immunological mediators. The patient may have been previously exposed to malaria. The short delay between infection and onset of neurological symptoms may be attributed to this immunological mediated damage following a memory immune response.

A drug-induced polyneuropathy is unlikely as

chloroquine and primaquine which this patient received are not known to produce polyneuropathy either by direct toxicity, idiosyncrasy or a toxic contaminant.⁷

The Guillain-Barré syndrome is an immunologically mediated, acute inflammatory demyelinating polyneuropathy occurring in all parts of the world at all ages. It has been previously reported following immunization,⁸ surgical operations, upper respiratory tract infections, psittacosis, *Mycoplasma pneumoniae*⁹ and viral infections such as cytomegalovirus, Epstein-Barr, varicella, measles, mumps and hepatitis.¹⁰

Recently a case of sarcoidosis presenting as an acute Guillain-Barré syndrome has also been reported.¹¹

As with most patients with uncomplicated Guillain-Barré syndrome, the patient in the present investigation also showed signs of recovery after 3 weeks. To my knowledge this is the first report of the Guillain-Barré syndrome following malaria.

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